LEFT-HEART BYPASS

by

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I SPEAK for Professor Ake Senning, Dr. Eric Carlens, Dr. S. A. Wesolowski, Dr. D. P. Hall, and our many collaborators both in Stockholm and in Brooklyn.

Mortality in patients with myocardial infarction and shock has been reported to be in excess of 84 percent by many authors, among them Biörck,[2] Wahlberg,[16] and Malach and Rosenberg.[10] The latter authors found 25 patients in 1 year at Kings County Hospital with myocardial infarction and blood pressures of 70 mm Hg (systolic) or lower, or with blood pressures more than 70 mm Hg below those known to exist before the coronary occlusion, accompanied by signs of peripheral vascular collapse. These patients all died, regardless of the type of therapy employed.

Upon reflection, the course of events in these patients is seen to be local ischemia, leading to poor myocardial function, giving rise in turn to a reduction in cardiac output and, secondarily, in systemic blood pressure. Secondary to this, in turn, is poor perfusion of the still-unoccluded portions of the coronary arterial tree and therefore a reduced possibility of survival of marginally arterialized muscle. Quite apart from the cardiac aspects of this chain of events, there is poor perfusion of the viscera generally, with secondary development of irreversible shock. Any measures likely to lead to survival in these patients not only must provide a greater head of pressure for myocardial perfusion through the remaining patent arterial channels, but must perfuse the viscera sufficiently to permit recovery from the endotoxin shock that accompanies the cardiac disaster. The presence of endotoxin in the peripheral blood when the blood pressure is low has been established by Nelson and Noyes,[12] and the enhancement of survival in endotoxin shock by left-heart bypass has been demonstrated by D. P. Hall and associates.[9]

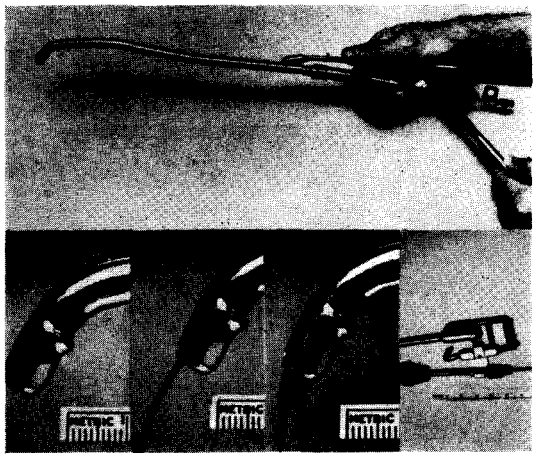

FIG. 1. The transseptal cannula for use in dog or man. *Top:* the entire cannula. *Bottom:* left, closeup of smooth tip of cannula; left center, closeup of tip with aspirating needle advanced as for confirmation of position; right center, closeup of tip with cutting lip and needle advanced; right, closeup of lever mechanism for fingertip advancement of cutting lip. (In part from Dennis, C., E. Carlens, A. Senning, D. P. Hall, J. R. Moreno, R. R. Cappelletti, and S. A. Wesolowski. Clinical use of a cannula for left heart bypass without thoracotomy. *Ann. Surg. 156*:623–637, 1962. Reprinted by permission of J. B. Lippincott Co.)

When Senning, Hall, Moreno, and I began to work in Stockholm in 1960, Senning and I had already resolved to pursue the physiological and technical problems of left-heart bypass. Our reasons lay, first, in the excessive blood trauma involved in prolonged use of an oxygenator if heart-lung bypass were to be undertaken, and second, in our being impressed by the limited volume flows of veno-arterial pumping without oxygenation (Connolly *et al.*[4]) and the disastrous cardiac response to the entrance of unsaturated blood into the coronary system in the presence of heart failure (Patt and associates[13]). Finally, we were not yet ready to pursue counterpulsation as Clauss *et al.*[3] had done. We were aware of Salisbury's suggestion that left-heart bypass might surpass heart–lung bypass for this purpose.[14]

Senning had observed benefit from partial left-heart bypass during the 15 to 45 min after open-heart repair of certain cardiac defects with the use of coronary flow interruption. Inasmuch as Bevegard and his associates had shown the feasibility of atrial septal puncture via the jugular vein for diagnostic purposes,[1] Senning proposed that we use the welcome sabbatical leave of one of us (C. D.) to work out jointly the problems of jugular, transseptal, left atrial cannulation without thoracotomy and to settle the question of diminution of metabolic work of the heart by such bypass.

Exploration of the interior of the atria in a series of cadavers showed that the anterior superior corner of the fossa ovalis provides the one consistently safe point of puncture into the left atrium. Fortuitously, the limbus of the fossa ovalis is one of the most striking landmarks to palpation, and in appreciation of this our group was joined by Carlens in perfecting the cannula (AB Stille-Werner, Stockholm, Sweden*) by which consistent puncture at the desired site has been possible without fluoroscopy (Fig. 1).

* In the U.S., available from Ohio Chemical Co., Madison, Wisconsin.

The placement of the cannula in a patient requires him to be supine with the head thrown slightly back and to the left. General anesthesia with tracheal intubation is most commonly used. The jugular vein is most easily exposed by a vertical incision between the sternal and clavicular heads of the sternomastoid muscle just above the inferior belly of the omohyoid, well above the entrance of the numerous low cervical tributaries. The cannula is filled with isotonic saline solution, lubricated with a small amount of thin mineral oil, and passed downward into the chest. When the tip has been advanced to about 10 cm below the top margin of the clavicle, the cannula is rotated so the tip is directed posteriorly and perhaps 15° medially, following which, with light pressure in that direction, the tip is passed caudally until it is felt to fall into the fossa ovalis. This occurs at a mean distance of 13 cm below the top margin of the clavicle (standard deviation from the mean of 13 cm is 1.3 cm, regardless of adult stature). In case of doubt, the cannula can be passed farther down and withdrawn until the arrest of the cannula tip by the limbus of the fossa ovalis is appreciated. In the dog the relative distance below the clavicle is usually greater than in man, and the site of puncture is usually found to lie almost precisely at the midsternal level. Fluoroscopy is no longer used in either man or dog.

The external circuit is of ½-in. tubing and includes a flexible siphonage chamber to avoid blood–air contact and a pump for return to the femoral artery (Fig. 2). In the clinical circuit, a heat exchanger constitutes the tubing leading to and from the patient and a flowmeter is included. Priming volume is 650 ml, and maximum flow is 5 liters/min.

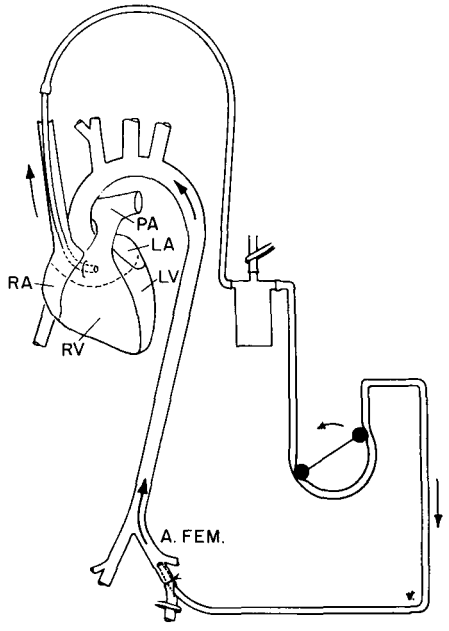

FIG. 2. The extracorporeal circuit. (Figures 2 and 3 in this presentation from Dennis, C., D. P. Hall, J. R. Moreno, and A. Senning. Left atrial cannulation without thoracotomy for total left heart bypass. *Acta Chir. Scand. 123*:267–279, 1962. Reprinted by permission of the publisher.)

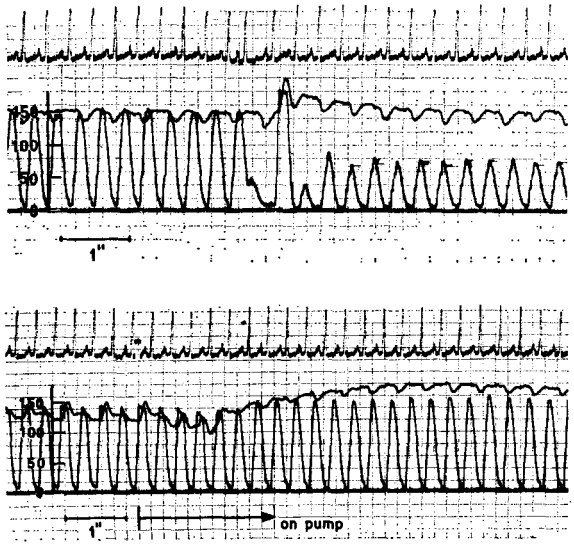

FIG. 3 (a) and (b). Pressure tracings during institution of closed-chest left-heart bypass.

Totality of diversion of blood from the left atrium has been demonstrated with pressure recordings and angiographic studies. Figure 3 shows a set of pressure tracings recorded while a dog was being placed upon left-heart bypass without thoracotomy. In Fig. 3a, aortic pressure rose within a few seconds to levels above the peak left ventricular pressure. In Fig. 3b, 32 sec after the start of bypass, an extrasystole emptied for the left ventricle, after which left ventricular pressure remained far below aortic. Figure 4 shows such a tracing upon termination of a period of total bypass by this circuitry, but with the chest open to permit the recording of pressures in several chambers. Here it is obvious that the left atrial pressure was lowered by left-heart bypass and that, upon cessation of bypass, ventricular filling became possible only as the left atrial pressure rose, secondary to termination of siphonage from this chamber. Note the small pulse imposed by the pump in the aortic tracing and the absence of the steep rise mentioned by Dr. Rainer.

The angiocardiographic evidence of totality of left-heart bypass without thoracotomy is presented in Fig. 5. As the figure indicates, after injection of dye into the pulmonary artery, returning blood was totally withdrawn from the left atrium, so that none entered the ventricle. Good perfusion occurred in retrograde fashion through the aorta (d, e, f), so that both coronary and cephalic vessels were well opacified (f).

The group of us has reported reduction of oxygen utilization on total left-heart bypass by approximately 50 percent, with proportional decreases on lesser degrees of bypass.[7] We have also reported long-term survival of nine out of 10 dogs after near-total bypass lasting 24 hr.[8] The sites of

puncture had healed completely in both of two dogs later held for 3 months before sacrifice. In these dogs there was an average plasma hemoglobin level below 80 mg percent at the end of bypass, and, in contrast to Dr. Rainer's observations, no drop in pH.

It is appropriate to mention here an experiment performed by the group of us in Stockholm, which has not been published in full. We undertook to see how long we could maintain a dog on nearly total left-heart bypass. As had been the case in the 10 dogs subjected to 24-hr total or nearly total left-heart bypass without thoracotomy, this dog did nicely for the first 24 hr. Thereafter, beginning at about 36 hr, he began to gain weight at the expense of the blood transfusion required to maintain a satisfactory blood volume. By the end of 83 hr of continuous bypass, he had developed enough edema to increase his body weight by 33 percent. The edema was largely centered about the shoulders, neck, and jowls, so that he more nearly resembled a Cheshire pig than a shepherd dog. In the light of this observation, our laboratory is beginning experiments to determine the role of pulsatile aortic pressure in lymphatic flow. Wesolowski *et al.* demonstrated this to be negligible for 4-hr perfusions a decade ago,[17] but the more prolonged support we are now considering would appear to require re-evaluation of this factor.

On the basis of the observations I have mentioned, our thesis is that both shock secondary to coronary arterial occlusion and left-heart failure due to valvular disease other than aortic insufficiency may well be ameliorated by left-heart bypass without thoracotomy. The following established effects of left-heart bypass are factors in this thesis: (1) maintenance of normal systemic blood pressure, and therefore proper perfusion of all tissues except that portion of the myocardium rendered ischemic by coronary occlusion; (2) relief of pulmonary edema and attendant arterial unsaturation by reduction of left atrial pressure to normal or below normal, repeatedly demonstrated in clinical patients; (3) reduction of the oxygen need of the myocardium, including the marginal myocardium rendered only partially ischemic by the coronary block, and therefore expected salvage of more muscle than without support; and (4) elevation of the oxygen tension in

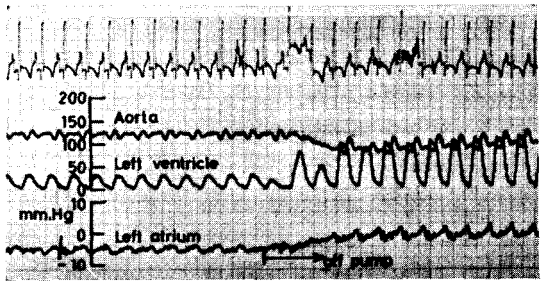

FIG. 4. Pressure tracings in a dog as open-chest left-heart bypass is terminated. (From Dennis, C., D. P. Hall, J. R. Moreno, and A. Senning. Reduction of the oxygen utilization of the heart by left-heart bypass. *Circulat. Res. 10*:298–305, 1962. Reprinted by permission of the American Heart Association, Inc.)

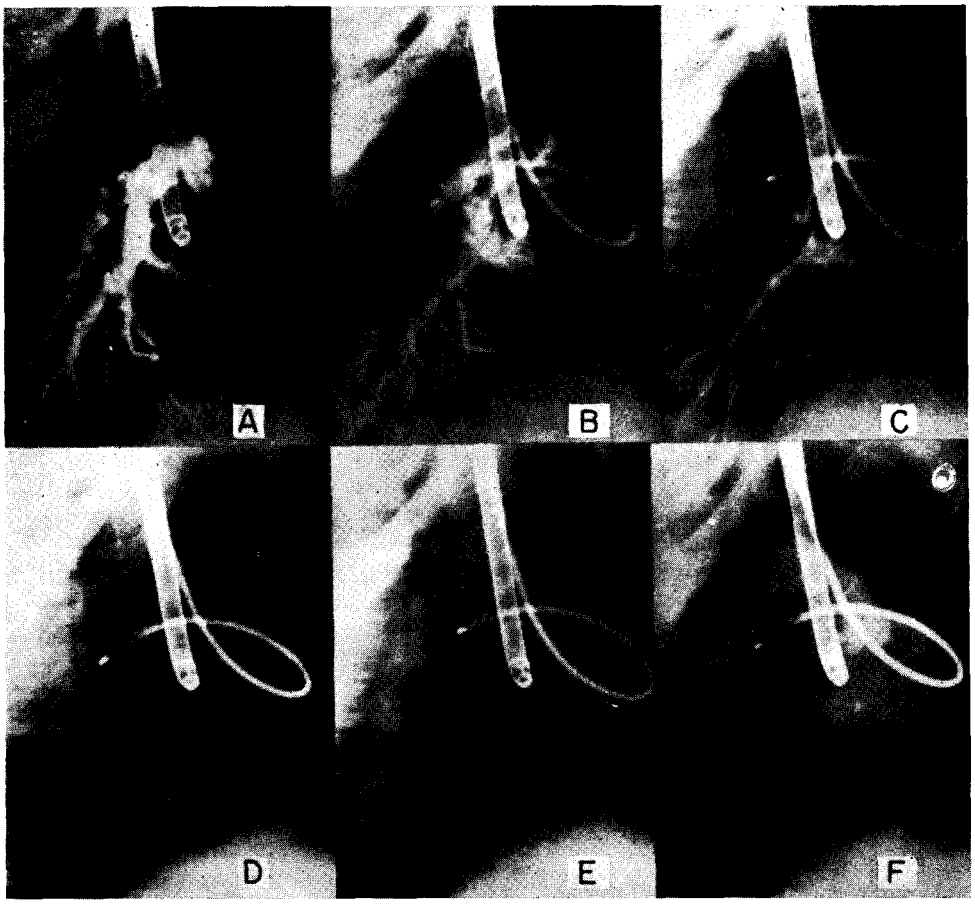

FIG. 5. Angiographic evidence of totality of left-heart bypass without thoracotomy—a series of films taken during 24 sec after injection of 50 ml of Urografin by catheter into the pulmonary artery. (From Moreno, J. R., A. Senning, C. O. Ovenfors, C. Dennis, and D. P. Hall. Angiographic studies during total left-heart bypass without thoracotomy. *Acta Radiol.* 57:17–23, 1962. Reprinted by permission of the publisher.)

the myocardium, indicated by a rise in coronary sinus oxygen saturation on institution of either partial or total left-heart bypass.

Our clinical experience is summarized in Table 1. The developmental errors of the first four cases listed and the tardy arrival of the next four need little comment and are failures not likely to recur as more experience and collaboration are gained (Table 2). Among the last four listed in the table, Case 12 was a man with a 10-day-old infarction, a duration not recognized until autopsy, in whom massive hemopericardium apparently began some 12 hr after institution of bypass. Improving circulation and urine formation and occasional disappearance of heart block had provided real encouragement until that time, when a rising venous pressure and per-

TABLE 1: *Left-Heart Bypass*

Serial no.*	Lesion	Duration of bypass, hr	Immediate result	Comments
9	0	5.5	Died	Collapse 4 days after fem. hernia c. gangrene of gut; stress ulcer, no infarct
10	0	4	Died	Dx error; massive pulmon. embolism
2	?	2	Died	False passage with primitive cannula
4	?	1	Died	Attempted under local anesthesia; vomited, drowned
7	?	0	Died	Cardiac massage 2 hr before arrival
6	?	0	Died	Cardiac massage 1 hr before arrival
11	LADC and Circ. aa.	5	Died	4x vent. fibrill. before arrival; massage during cannulation
5	LMC	4	Died	Fibrill. before and during cannulation (open massage)
12	LADC and PDCA	17	Died	Hemopericardium; path. 10-day-old infarct with early aneurysm
3	Mitr. insuff. pulm. edema	16	Good	Congestion cleared and circ. stable without pump; Starr valve placed later
1	Infarct sept. and post. wall	16	Imp.	After 12 hr pt. cooled and VSD sutured; died of failure of septal closure
8	Infarct sept. and post. wall	7	Imp.	Awoke stabilized; cannulas removed 2 hr later; died of arrest ½ hr thereafter

* Case 1: Dennis, Senning, Hall, and Moreno. Case 2: Dennis, Hall, and Moreno. Case 3: Hall and Ellison, Augusta, Ga. Cases 6, 7, and 8: Carlens and Ekestrom, Stockholm. Cases 4, 5, 9, 10, 11, and 12: Brooklyn.

sistence of complete atrioventricular dissociation led us to continue bypass. Hemopericardium was not recognized, and he was given up after 17 hr of bypass. An already developing ventricular aneurysm might have made pericardiocentesis hazardous. Case 3 suggests that left-heart bypass should be thoroughly evaluated in patients with mitral disease who cannot securely be prepared for definitive repair in other ways, for she regained cardiovascular stability and enjoyed several hours of respite between cessation of bypass and beginning of thoracotomy for placement of the Starr valve, only to die later of pulmonary hypertension.[5] Our near success in Case 1, also, offers hope that definitive repair of some complications of myocardial infarction may be feasible in the acute state.[5] Finally, Case 8 suggests the hope that some patients with myocardial infarction may respond sufficiently to nothing more than temporary support, and survive, as did our first myocardial-infarction patient placed on heart-lung bypass, presented by Dr. Stuckey. Continuous monitoring with electrodes and direct-current converter in place might have been successful in this instance.

TABLE 2: *Diagnostic and Developmental Errors in Left-Heart Bypass Cases*

Serial no.	Diagnostic errors	
9	Collapse from postoperative bleeding; stress ulcer	1
10	Massive pulmonary embolism	1
	Developmental errors	
2	False passage with primitive cannula	1
4	Local anesthesia, vomiting and drowning	1
5, 6, 7, 11	Arrived too late and cannulation done during and after prolonged cardiac massage	4
12	Hemopericardium after 10-hr bypass	1
3	Mitral insufficiency and congestion cleared by 16-hr bypass; died of pulmonary hypertension after uneventful placement of Starr valve	1
1	Good circulation restored and postinfarction VSD repaired; died of breakdown of repair	1
8	Stabilized on 7 hr of bypass; died 2½ hr later of sudden arrest	1

At least three of the cases had sufficiently localized coronary occlusion that hypothermia, coupled with addition of right-heart bypass, such as that employed in Case 1 for repair of a ventricular septal defect, might have been used for definitive plastic repair of the coronary arterial tree. Proper diagnostic methods should make this feasible.

In conclusion, left-heart bypass without thoracotomy is possible within an hour of the decision to use it, and is theoretically sound. With the increased collaboration now developing with our confreres in cardiology, anesthesiology, and radiology, it should be possible either (1) to provide temporary support for those in whom it would suffice; (2) to provide coronary arterial plastic repair for those with sufficiently localized occlusions and early enough in their attacks to have still viable muscle; (3) to excise the infarct where it is necessary in later cases with localized infarction; or (4) to exclude those for whom stage and extent of disease make survival impossible.

ACKNOWLEDGMENTS

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